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Research Paper

Periprosthetic wear particle migration and distribution modelling and the implication for osteolysis in cementless total hip replacement

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ABSTRACT

In total hip replacement (THR), wear particles play a significant role in osteolysis and have been observed in locations as remote as the tip of femoral stem. However, there is no clear understanding of the factors and mechanisms causing, or contributing to particle migration to the periprosthetic tissue. Interfacial gaps provide a route for particle laden joint fluid to transport wear particles to the periprosthetic tissue and cause osteolysis. It is likely that capsular pressure, gap dimensions and micromotion of the gap during cyclic loading of an implant, play defining roles to facilitate particle migration. In order to obtain a better understanding of the above mechanisms and factors, transient two-dimensional computational fluid dynamic simulations have been performed for the flow in the lateral side of a cementless stem-femur system including the joint capsule, a gap in communication with the capsule and the surrounding bone. A discrete phase model to describe particle motion has been employed. Key findings from these simulations include: (1) Particles were shown to enter the periprosthetic tissue along the entire length of the gap but with higher concentrations at both proximal and distal ends of the gap and a maximum rate of particle accumulation in the distal regions. (2) High capsular pressure, rather than gap micromotion, has been shown to be the main driving force for particle migration to periprosthetic tissue. (3) Implant micromotion was shown to pump out rather than draw in particles to the interfacial gaps. (4) Particle concentrations are consistent with known distributions of (i) focal osteolysis at the distal end of the gap and (ii) linear osteolysis along the entire gap length.

1. Introduction

Osteolysis, the biological reaction to wear debris, occurs in both cemented and cementless total hip replacement. Osteolytic lesions are particularly more extensive and common in cementless femoral components when there is a lack of seal in the bone–implant interface (Harris, 1994). In radiographs, osteolytic lesions can appear as focal scallop shaped

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radiolucencies at parts of the interface or as linear radiolucencies that extend along the edge of an implant (Freeman, 1999). It has been observed that the scallop shaped lesions are not isolated and are contiguous with linear lesions (Iwaki et al., 2002) suggesting a path of communication between them (Iwaki et al., 2002), which may facilitate particle migration to that region.

In a replaced joint, particles are constantly being released to the joint capsule due to wear of the bearing surfaces. This leads to accumulation of particles in the effective joint space. There is evidence that shows particles migrate deep into the tissue (Revell et al., 1997; Bauer and Schils, 1999; Goodman et al., 2009) and travel along smooth surfaces of non-circumferentially coated stems where there is no interlock between the bone/fibrous tissue and the metal surface of the implant (Bobyn et al., 1995). Furthermore, the interfacial gaps, which are in communication with the effective joint space, provide a conduit for wear particles and other implant byproducts, and transmits waves of fluid pressure during cyclic loading of the prosthesis (Goodman et al., 2009). It is not only the presence, but the size and the concentration of particle, determined predominantly by the polyethylene (PE) wear rate, that play a role in osteolysis development (Gallo et al., 2010; Goodman et al., 1990).

There are numerous studies indicating a relationship between the presences of the wear particles and osteolysis in the periprosthetic tissue. It has been shown that polyethylene, in particle form, induces an inflammatory reaction, including the infiltration of macrophages, foreign-body giant cells, and fibrous tissue similar to that seen around loose implants (Goodman et al., 1988). Studies performed with use of a bone-chamber model have also shown that bone ingrowth is reduced in the presence of particulate degradation products (Goodman et al., 1995).

Revell et al. (1997) showed that polyethylene particle concentration retrieved from different periprosthetic sites ranged between 5.2×10^{8} and 9.17×10^{10} particles per gram of tissue. Sites containing more than a critical value of approximately 1×10^{10} particles per gram of tissue had developed focal osteolysis. This critical threshold was also confirmed by Kobayashi et al. (1997). There is also strong evidence that correlates the presence of polyethylene particles with osteolysis generation (Orishimo et al., 2003; Schmalzried et al., 1992; Maloney et al., 1990; Willert et al., 1990). Some of these osteolytic lesions are located far from the joint capsule where particles are generated (Schmalzried et al., 1992). To explain the migration of particles deep into the tissue and to locations as remote as the tip of the femoral stem, the concept of 'effective joint space' has been postulated (Schmalzried and Callaghan, 1999). This concept proposes that particles may migrate to all regions to which joint fluid has access, including interfacial gaps which may extend along the entire length of a femoral stem. However, the mechanisms facilitating such migration are not clearly understood. Furthermore, there is a lack of descriptive detail on the extent of particle penetration into, and their distribution pattern in the periprosthetic tissue in cementless implants.

Two possible transport mechanisms of convection (i.e. hydraulic transfer) and cellular transport have been suggested by Elfick et al. (2003). In the former, particles are carried with the fluid flow through the path of least resistance in the periprosthetic tissue as the result of hydrodynamic forces generated by pressure differences. Two main sources that may generate hydrodynamic forces to drive particles in the effective joint space may be capsular pressure (Schmalzried et al., 1997; Robertsson et al., 1997) and implant micromotion (Bartlett et al., 2008). Wear particles may stimulate the joint lining membrane to produce fluid leading to excessive effusion, which in turn increases joint hydrostatic pressure (Goodman et al., 2009; Gallo et al., 2013). It has been shown that in problematic THRs in which the joint capsule experiences excessive effusion (Wingstrand et al., 1990; Hendrix et al., 1983; Tarasevicius et al., 2007) and soft tissue fibrosis (Robertsson et al., 1997), the capsular pressure may increase significantly, particularly during physical activities such as stair climbing (Hendrix et al., 1983). In addition, when a stem is under physiological loading, the difference between bone and metal stiffness causes different degrees of deformation in these two materials, which in turn leads to micromotion at the interface. This micromotion may cause the interfacial gaps to open and close during physical activity, creating a mechanism which may also pump the particle laden joint fluid into and out of an interfacial gap.

In a previous study (Alidousti et al., 2011), it was proposed that capsular pressure, gap dimension and micromotion may play a defining role in generating periprosthetic flows and the extent of the influence of each of these parameters in generating high fluid pressure and velocity in the gap and surrounding bone was investigated. In the current study, in order to investigate the effect of these parameters on particle migration to the periprosthetic tissue, a discrete phase model (DPM) representing the particles as a second phase, is added to the 2D computational fluid dynamic (CFD) models developed by Alidousti et al. (2011) and the motion of particles in the effective joint space is simulated.

2. Method

2.1. *Model construction*

The geometry of the simulations was derived from a cross-sectional cut of an anatomically realistic model of an implanted femur and the surrounding bone. It consisted of an interfacial gap located in the lateral side of a replaced joint which was in communication with the joint capsule and its surrounding porous bone (cf. Fig. 1). The models constructed from this geometry included gaps with different lengths and widths and the implant wall was prescribed with an opening and closing motion, pivoting around the bottom of the gap, to represent implant micromotion during a gait cycle (cf. Fig. 1). The choice of gap dimensions and displacements for the current study (cf. Table 1) is described in detail by Alidousti et al. (2011).

A gait cycle during which micromotion occurs was presented with a truncated period of $T=0.2$ s. This time period only represents the instances in which forces are acting on the femoral stem (cf. Alidousti et al. (2011) for more details). The same initial peak load during gait has been calculated by Kurtz et al. (2005). The models were transient and displacement-driven